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in this study, we identified 15 susceptibility loci associated with covid-19 across european ancestries, including three novel loci associated with critical illness. we identified nine susceptibility loci across european ancestries that were associated with reporting an infection, including the newly discovered 13q21.31 locus, as well as loci previously associated with type i interferon response and susceptibility to several types of infections, which overlap with ours in their linkage to the 2q14.1 and 2q13 loci. we identified four loci associated with severe covid-19 outcomes, which independently replicated in the two largest covid-19 genome-wide association studies to date. we verified a strong trans-ancestry signal at 6p21.3 and examined the presence of association between the loci of interest and covid-19 severity, reported infection, and severity of influenza. we demonstrated that two previously identified loci associated with severe influenza were also associated with severe covid-19 and that these signals persisted across the ancestries included in our study. we identified a signal at the 3p21.31 locus in multiple european populations that significantly improved model fit compared to the 2q14.1 locus, which was previously reported as the strongest signal of covid-19 severity and that we corroborated in african american and mexican populations. we found a missense mutation in the dcaf16a gene and two rare dcaf16a loss-of-function variants in individuals with severe covid-19 and replicated the association with severe influenza, indicating that rs1339894 could be a causal variant at the 3p21.31 locus associated with the severity of covid-19.

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we defined a locus as replicated when we discovered the same covid-19 locus previously identified in another independent study (ie,, where genome-wide significant variants in a particular region have been consistently reported by different groups). for sars-cov-2 infection, we used four independent studies with available genome-wide significant variants that had corresponding genome-wide significant loci in our study (supplementary table 5). epidemiological evidence (e.g. changes in surgical procedures or public health interventions) can lead to an increased proportion of testing positive for covid-19, even in an otherwise healthy cohort of individuals, which may result in an apparent positive genetic correlation with disease that is not actually due to shared etiology. to avoid this confounding effect, we focused on the genetic correlation between covid-19 and the lung (i.e. an important site of sars-cov-2 infection and clinical presentation of the disease) and computed the genetic correlation in a null model that does not include any confounding factors. the attenuation of genetic correlations with the lung due to confounding is illustrated by the magnitude of the genetic correlation of our reported most strongly associated locus at dpp9 with the lung, which drops from 0.35 before to 0.24 in the control and 0.16 in the null model. notably, several of our top genetic correlations involved a disease with differing epidemiology; for example, we observed a genetic correlation of 0.36 between covid-19 and inflammatory bowel disease (e. crohn’s disease), whose epidemiology differs from that of covid-19. the genetic correlation of 0.47 between covid-19 and pregnancy loss and twinning (previously reported 36) is also likely due to confounding by co-infection of mothers and fetuses during pregnancy; the molecular and cellular phenotypes in the fetus might be more similar to covid-19 than those during the gestation period. 5ec8ef588b

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